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Award Number: DAMD17-03-2-0024

TITLE: Physiologic and Endocrine Correlates of Overweight and Obesity in African Americans and Caucasians

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REPORT DATE: March 2007

TYPE OF REPORT: Annual

PREPARED FOR: U.S. Army Medical Research and Materiel Command
Fort Detrick, Maryland 21702-5012

REPORT DOCUMENTATION PAGE

Form Approved
OMB No. 0704-0188

1. REPORT DATE *(DD-MM-YYYY)*	**2. REPORT TYPE**	**3. DATES COVERED** *(From - To)*
01-03-2007	Annual	01 Mar 03 – 28 Feb 07

4. TITLE AND SUBTITLE
Physiologic and Endocrine Correlates of Overweight and Obesity in African Americans and Caucasians

5a. CONTRACT NUMBER

5b. GRANT NUMBER
DAMD17-03-2-0024

5c. PROGRAM ELEMENT NUMBER

6. AUTHOR(S)
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5d. PROJECT NUMBER

5e. TASK NUMBER

5f. WORK UNIT NUMBER

7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)
Henry M. Jackson Foundation for the Advancement of Military Medicine
Rockville MD 20852

8. PERFORMING ORGANIZATION REPORT NUMBER

9. SPONSORING / MONITORING AGENCY NAME(S) AND ADDRESS(ES)
U.S. Army Medical Research and Materiel Command
Fort Detrick, Maryland 21702-5012

10. SPONSOR/MONITOR'S ACRONYM(S)

11. SPONSOR/MONITOR'S REPORT NUMBER(S)

12. DISTRIBUTION / AVAILABILITY STATEMENT
Approved for Public Release; Distribution Unlimited

13. SUPPLEMENTARY NOTES

14. ABSTRACT:
Obesity has reached epidemic levels and yet the incidence continues to rise. The current study is seeking to examine the hypothesis that obesity may reflect dysfunctioning of the hypothalamic-pituitary-adrenal (HPA) axis in response to stressors. African American persons are at greatest risk, but reasons for this difference are unknown. We will study 120 men and women of Caucasian and African American ethnicity and examine their responses to physiologic stressors: exercise and ingestion of a meal. Methods: The HPA axis will be studied in some detail by using two stressor paradigms and two steroid regimens. We expect to be able to detect subtle differences in HPA axis reactivity in obese individuals that might contribute to morbidity and perhaps even make individuals resistant to therapeutic interventions. Results: We have enrolled 124 participants, with 93 completed. Data collection and analyses are proceeding on schedule. Two abstracts were presented in 2006 and one is submitted and accepted for presentation in Summer 2007. Conclusions: We are on schedule for all study milestones and look forward to being able to answer the important questions regarding the potential role of the HPA axis in obesity.

15. SUBJECT TERMS
obesity, insulin sensitivity, glucocorticoids, metabolic syndrome, African Americans, Caucasians, exercise, meal feeding

16. SECURITY CLASSIFICATION OF:			**17. LIMITATION OF ABSTRACT**	**18. NUMBER OF PAGES**	**19a. NAME OF RESPONSIBLE PERSON**
a. REPORT	**b. ABSTRACT**	**c. THIS PAGE**			USAMRMC
U	U	U	UU	13	**19b. TELEPHONE NUMBER** *(include area code)*

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INTRODUCTION

Obesity has reached epidemic levels and yet the incidence continues to rise. The current study is seeking to examine the hypothesis that obesity may reflect a dysfunction of the hypothalamic-pituitary-adrenal (HPA) axis in response to stressors. African American persons are at greatest risk, but reasons for this difference are unknown. We are studying 120 men and women of Caucasian (CA) and African American (AA) ethnicity to examine their responses to physiologic stressors: exercise and ingestion of a meal.

BODY

Year Four:

1. *Recruit, screen, and test 10 Overweight/Obese and 10 Non-obese subjects*

Table 1. Breakdown of Normal/Overweight/Obese Participants by Ethnicity (Year 4)

Normal (NW) Overweight (OW) & Obese (OB)	CA-NW	AA-NW	CA-OW	AA-OW	CA-OB	AA-OB
Screened	12	29	3	29	8	32
Recruited/Enrolled	3	8	3	9	2	12
In Progress	0	1	0	0	1	3
Dropped	1	1	0	2	0	4
Completed	**2**	**6**	**3**	**7**	**1**	**5**
Total Screened	**N = 124**					
Total Enrolled	**N = 37**					
Total Completed	**N = 24**					

2. *Complete subject recruitment*

Since the start of the study, we have recruited 124 subjects. Of those, 93 subjects have completed the study and 5 are in progress; 26 have dropped out (Table 2). Thus, we have less than 30 subjects to recruit (20 African Americans for the normal and obese group and 7 Caucasian obese subjects). We have asked for an increase in the number of subjects we can recruit due to the larger dropout rate among African American subjects (n = 24 AA and n = 2 CA). Enrolling subjects into the obese category is difficult due to our inclusion/exclusion criteria. For example, some are currently taking blood pressure or cholesterol lowering medications and many persons who call to participate have BMIs above our cutoff.

In the last year, we provided body composition services and recruited at the Healthy You Health and Fitness Expo in Silver Spring, MD in September 2006. The same service was performed at the Montgomery County Heart Health Symposium for African Americans in Germantown, MD in March 2006, both as recruitment strategies. We expect to finish recruitment within the next few months.

Table 2. Years 1-4 Subject Recruitment and Progress

		Normal	Overweight	Obese	Total
AA	Enrolled	22	33	25	80
	In Progress	1	0	3	4
	Completed	13	24	15	52
	Dropped	8	9	7	24
	Needed	11	0	9	20
CA	Enrolled	18	16	10	44
	In Progress	0	0	1	1
	Completed	16	16	9	41
	Dropped	2	0	0	2
	Needed	0	0	7	7
	Total In Progress	5			
	Total Completed	93			
	Total Needed	27			

3. *Complete subject testing*

To date, 93 subjects have completed testing in the laboratory; 5 subjects are in progress and are scheduled to finish testing soon. We still need to recruit and complete 27 subjects. Refer to Table 2.

4. *Continue evaluating, reducing, and analyzing data*

Multiple meetings have occurred among the PI, Co-investigators, with the Project Coordinator and other key staff on a regular basis to discuss issues and examine data collected on all completed subjects. Preliminary hormone, psychological, and other physiological data have been analyzed while preparing for three different abstracts and an oral presentation that were presented or are to be presented at conferences this year. We are ahead of schedule for this goal.

5. *Continue with biochemical analyses.*

Radioimmunoassay (RIA) analyses are in process for several of the different hormones of interest. Specifically, we have competed ACTH data on 61 subjects, Insulin data on 83 subjects, Cortisol (CORT) data on 23 subjects, and DHEA data on 43 subjects. Unfortunately, we have had the same problem with our new CORT kits that we experienced last year. The manufacturer stopped production and we had to find yet another manufacturer for the second time. This caused us to start from the beginning and had to repeat testing on 21 subjects. We have found a new manufacturer for the ACTH kits and progress has been made to catch up with the delays. Currently, CORT and ACTH are being run simultaneously at a collaborative laboratory at NIH. We plan to continue the biochemical analyses on Insulin, CORT, ACTH, and DHEA hormones. This goal is on track.

6. *Begin statistical analyses on ethnicity/obesity and potential interactions.*

We have partial data from 98 subjects (93 have complete data and 5 have partial data). From preliminary data analyses, weight, BMI, maximal aerobic capacity, and fasting blood glucose were significantly different across weight groups. Weight, BMI, and fasting blood glucose were not significantly different between AA and CA. However, despite similar weights and BMI, maximal aerobic capacity (VO_{2max}) was significantly lower in AA than CA (Table 3). Overall, VO_{2max} was 44.4 ± 8.5 (ml/kg/min) for CA and 37.6 ± 10.1 (ml/kg/min) for AA.

Table 3. Characteristics and Physiological Measures by Ethnicity and Weight Group

	CA-NW (n=16)	CA-OW (n=16)	CA-OB (n=10)	AA-NW (n=14)	AA-OW (n=24)	AA-OB (n=18)
Age (yrs)	24.0 ± 3.6	30.6 ± 3.8	30.5 ± 5.9	27.1 ± 7.1	30.2 ± 8.0	34.3 ± 6.9
Weight (kg)	62.6 ± 9.5	85.4 ± 8.7	105.2 ± 12.7	66.5 ± 9.0	83.4 ± 13.2	93.4 ± 14.6
BMI (kg/m^2)	22.8 ± 1.5	27.1 ± 1.4	33.6 ± 2.5	22.9 ± 1.4	27.5 ± 1.5	32.7 ± 2.2
VO_{2max} (ml/kg/min)*	50.3 ± 4.9	43.5 ± 8.3	36.4 ± 6.0	43.5 ± 9.5	40.2 ± 9.4	29.1 ± 5.2
Glucose (mmol/L)	5.1 ± 0.6	5.4 ± 0.7	5.5 ± 0.7	5.3 ± 0.9	5.2 ± 0.7	5.3 ± 0.7

Values are mean ± SD.

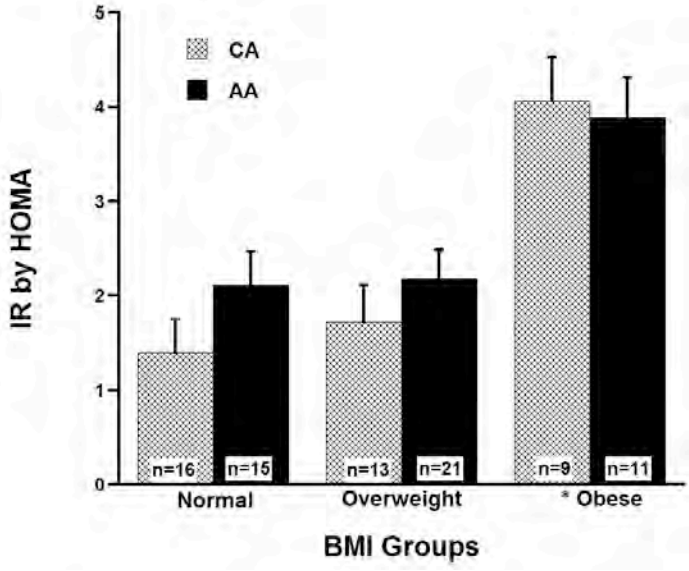
7. *Reduce and interpret data on HPA reactivity from the exercise and meal challenge tests as a function of ethnicity and obesity after all subjects have been tested.*

Areas under the curve for the exercise challenge and areas under the curve for the meal challenge were calculated for ACTH (n = 30 CA, 31 AA) and CORT (n = 10 CA, 15 AA) responses. Due to the problems with the RIA kits, most of our work had to be repeated and the results presented are based on a small number of subjects, especially CORT. Preliminary analyses of the data did not show an ethnic or weight group difference in ACTH or CORT production in response to the exercise challenge or the meal challenge across all treatments. Under treatment 2, both AA and CA had smaller increases in ACTH and CORT than under treatments 1 or 3. Preliminary trends show a difference in the magnitude and timing of peak ACTH responses to exercise. The peak in ACTH is smaller and seen earlier in AA than CA.

8. *Reduce and interpret data describing relationship between HPA axis resistance to feedback control and insulin resistance as a function of obesity and ethnicity after all subjects have been tested.*

Insulin resistance (IR) was calculated by the homeostasis model assessment (HOMA) method, which is obtained by multiplying fasting insulin (μIU/mL) and glucose (mmol/L) levels and dividing the product by a constant of 22.5. IR by HOMA (μIU/mL x mmol/L) was calculated for 47 AA and 38 CA (Figure 1). Preliminary analyses show that individuals with a higher BMI have a greater IR. Differences were found among BMI groups, with the OB group being significantly different than the NW and OW group. There were no differences in ethnicity.

Figure 1. Insulin Resistance by HOMA across BMI Groups



9. *Reduce and interpret data describing relation between exercise-associated increases in insulin and glucocorticoid sensitivity as a function of ethnicity.*

Areas under the curve (AUC) for insulin were calculated over the course of the meal challenge. Preliminary analyses of the data showed significant differences between AA and CA across all three treatment groups. AA produced more insulin than CA for every treatment condition (Table 4). Individuals were grouped by BMI (normal weight/NW: 18≤25, over weight/OW: 25≤30, and obese/OB: 30≤38). Significant differences across BMI groups were also noted for treatments 1 and 2, with the OB group releasing significantly greater amounts of insulin than NW and OW. When analyzed by ethnicity, significant differences in BMI groups were found only among CA, and that was with Treatment 1: OB CA produced more insulin than NW and OW CA.

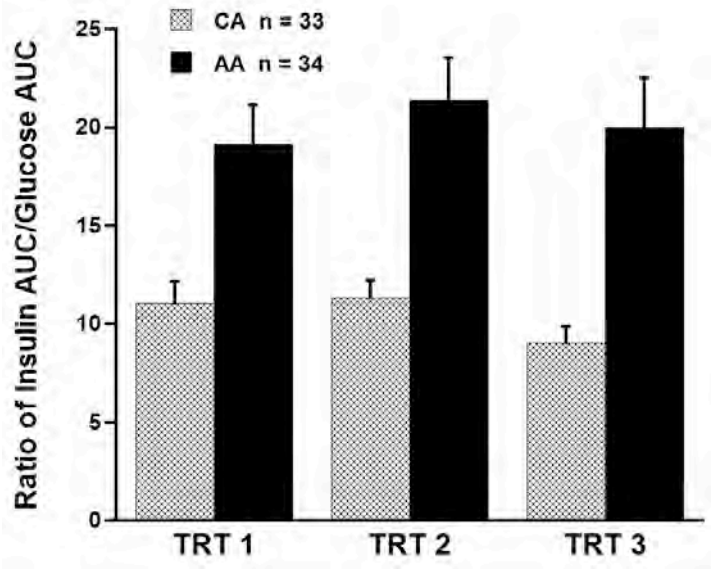
Table 4. Insulin AUC (μIU/mL/70min) by ethnicity and BMI

	n	TRT 1	TRT 2	TRT 3
AA *	36	9225 ± 996	12523 ± 1399	9863 ± 1240
CA	36	5609 ± 586	7230 ± 521	4374 ± 431
AA NW	9	9483 ± 1687	11575 ± 2029	7533 ± 1385
AA OW	17	7789 ± 1473	9883 ± 1876	9593 ± 2208
AA OB	10	11434 ± 2031	17598 ± 3002	12184 ± 1814
CA NW	14	5476 ± 827	7794 ± 721	4528 ± 422
CA OW	14	3805 ± 472	5860 ± 768	3335 ± 535
CA OB	8	* 8999 ± 1523	8466 ± 1300	5806 ± 1372

Values are mean ± SE. * $p < 0.05$.

Areas under the curve (AUC) for glucose were calculated over the course of the meal challenge. Preliminary analyses of the data showed no differences by ethnicity or BMI groups across all treatments (Data not shown). The ratio of AUC for insulin to glucose was calculated and significant differences were found across all treatments by ethnicity (Figure 2). Despite similar plasma glucose levels for AA and CA, AA released a larger amount of insulin in response to the same meal challenge. We are not attempting to interpret these data as the codes for treatment groups have not been broken.

Figure 2. Ratio of AUC for Insulin to Glucose across Treatments

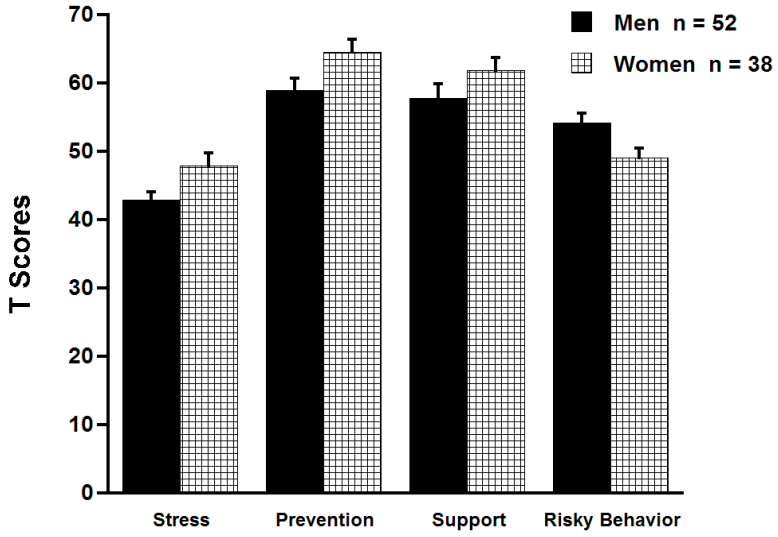


10. *Examine data as a function of gender after 20 men and 20 women have been tested.*

Expected gender differences were found in the preliminary data analyses between men and women. Men have a greater maximal aerobic capacity (VO_{2max}) (Men: 44.6 ± 1.2 vs. Women 36.1 ± 1.4 ml/kg/min), waist circumference (Men: 90.4 ± 1.6 vs. Women: 83.3 ± 1.9 cm), and a lower percent body fat (Men: 24.3 ± 0.8 vs. Women: 36.2 ± 1.1 %) than women. No differences were found in fasting glucose or insulin resistance as measured by HOMA IR.

Gender differences were also for select psychological variables on the Stress Profile and the Beck Depression Inventory (BDI) questionnaire. Although women had significantly greater scores for self-reported stress, they had higher scores for health prevention behaviors and a greater social support network (ns) than men (Figure 3). Men reported participating in risky behaviors (ARC) more frequently than women. Men also had significantly lower scores on the BDI (Men: 2.6 ± 0.4 vs. Women: 4.7 ± 0.8) as compared to women. The trend for greater coping strategies among women and the practice of preventive health strategies may offset the negative feelings of stress and depression in women.

Figure 3. Differences in Stress Profile Subscales



11. *Prepare report on results.*

This has not been prepared since an extension has been granted for the study until 31 March 2008.

KEY RESEARCH ACCOMPLISHMENTS

- No significant differences were found between AA and CA in the measurements of weight (AA: ± vs. CA: ± kg), BMI (AA: 28.0 ± 4.1 vs. CA: 26.7 ± 4.9 kg/m^2), waist circumference (AA: 88.5 ± 11.5 vs. CA: 85.7 ± 14.4 cm), and fasting plasma glucose (AA: 5.3 ± 0.8 vs. CA: 5.3 ± 0.7 mmol/L).
- Maximal aerobic capacity ($V0_{2max}$) was significantly lower in AA than CA (37.6 ± 10.1 and 44.4 ± 8.5 ml/kg/min for AA and CA, respectively) [$F_{(1,91)} = 12.8$, $p = 0.001$].
- Insulin Resistance (IR) by HOMA (μIU/mL x mmol/L) was significantly greatest in the Obese (4.0 ± 0.33) compared to Normal (1.8 ± 0.3) and Overweight (2.0 ± 0.3) AA and CA grouped by BMI [$F_{(2,79)} = 17.4$, $p < 0.001$].
- In AA, IR was positively correlated with stress ($r = 0.54$) and negative appraisal ($r = 0.54$). In CA, IR was positively correlated with BMI ($r = 0.70$), WC ($r = 0.68$), body fat ($r=0.49$), and social support ($r=0.40$), and negatively correlated with exercise ($r = -0.40$) and VO_2max ($r = -0.62$). In AA, increasing levels of stress and negative appraisal were associated with greater IR.
- AA had morning basal ACTH concentrations comparable to CA and cortisol concentrations that were significantly lower [$F_{(1,22)} = 4.45$, $p = 0.48$]. AA and CA had comparable ACTH responses to exercise and AA had significantly blunted cortisol responses [$F_{(1,22)} = 15.130$, $p = 0.01$]. Glucose levels after a liquid meal did not differ between AA and CA, but insulin

responses were significantly higher in AA [$F_{(1,75)} = 6.578$, $p = 0.012$; AUC -$F_{(1,75)} = 7.092$, $p = 0.009$].

- Women had significantly greater scores for self reported stress (Women: 47.9 ± 1.9 vs. Men: 42.9 ± 1.2) than men [$F_{(1,88)} = 5.206$, $p = 0.025$], but had higher scores for health prevention behaviors (Women: 64.5 ± 1.9 vs. Men: 59.1 ± 1.7) than men [$F_{(1,88)} = 4.533$, $p = 0.036$]. Men had significantly higher scores for participating in risky behaviors (Men: 54.2 ± 1.4 vs. Women: 49.0 ± 1.4) than women [$F_{(1,88)} = 6.275$, $p = 0.014$]. Men also had significantly lower scores on the Beck Depression Inventory (Men: 2.6 ± 0.4 vs. Women: 4.7 ± 0.8) as compared to women [$F_{(1,85)} = 5.480$, $p = 0.022$].

REPORTABLE OUTCOMES

ABSTRACTS 2006/07

1. Kim SJ, Oates C, Fendrick N, Zeno S, Faraday M, Sbrocco T, Poth M, and Deuster P. Physiological and Psychological Correlates of Insulin Resistance in African Americans and Caucasians. Presented at the Society for Behavioral Medicine 2006.
2. Fendrick N, Stephens Q, Oates C, Kim S, Zeno S, Faraday M, Poth M, and Deuster P. Physiologic and Behavioral Correlates of Obesity in African Americans and Caucasians. Presented at the 2006 DoD Military Health Research Forum (MHRF).
3. Kim SJ, Poth M, Deuster P, Fendrick N, and Zeno S. Differential Hormone Responses to a Liquid Meal and Exercise in African Americans and Caucasians. Accepted to present at the Endocrine Society 2007.
4. Stephens Q, Deuster P, and Poth M. Applicability of VO2 max Criteria in African American and Caucasian Individuals. Submitted to the American College of Sports Medicine.

MANUSCRIPTS

1. Stephens Q, Deuster P, and Poth M. Applicability of VO2 max Criteria in African American and Caucasian Individuals. Submitted to Health and Ethnicity
2. Oates C, Kim SJ, Faraday M, Stephens Q, and Deuster P. Physiological and Psychological Correlates of Insulin Resistance in African Americans and Caucasians. In preparation for Annals of Behavioral Medicine.

PRESENTATIONS

1. Stephens Q. Featured Guest Speaker – Montgomery County Cable Community Comments Television Program (Topic: Cardiovascular Disease and Exercise) in May 2006
2. Kim S. HPA Axis Responsivity and Ethnicity. Oral presentation for American Psychological Association meeting in New Orleans, LA in August 2006

GRANTS SUBMITTED

1. P20 NIH USH Health Disparities Research Center

CONCLUSIONS

Metabolic syndrome is a combination of cardiovascular risk factors including abdominal obesity, dyslipidemia, hypertension, insulin resistance, proinflammatory state, and prothrombotic state. Those with metabolic syndrome are at risk for other diseases including coronary heart disease, stroke, and Type 2 diabetes. Recent investigations have demonstrated that ethnic and

gender-based differences continue to exist in the prevalence, associated factors, and status of this disease.

Several socioeconomic variables have been identified as factors that are significantly associated with the metabolic syndrome. Low socioeconomic position (SEP) is associated with the prevalence of metabolic syndrome in AA and CA and the association is stronger in women than in men. Furthermore, low education, low poverty income ratio, and socioeconomic position were specifically related to the metabolic syndrome in women. Despite the gender differences that have been seen, education proves to be a variable associated with the components of metabolic syndrome. Improving access to higher education and targeting efforts at intervention and prevention towards low-SEP populations could reduce the risk for metabolic syndrome. There are racial differences in the prevalence of individual and clustered components of the metabolic syndrome. Specifically, the HTN component of the metabolic syndrome is significantly greater among AA with (73.1%) and without (47.5%) diabetes in comparison to their CA (58.6% and 32.4%) and Mexican American (50.8% and 23.4%) counterparts (with and without diabetes), respectively. Additionally, AA had a higher prevalence of elevated serum C-reactive protein and high plasma fibrinogen than CA. In diabetics, abdominal obesity is more prevalent among CA (80.6 %) than Mexican Americans (67.8 %).

Insulin resistance is more common in African Americans than Caucasians. However, a single bout of exercise has been shown to improve insulin sensitivity in sedentary overweight/obese African American women in response to a 75 min brisk treadmill walk. This response was also previously observed in sedentary CA individuals. Since it has established that insulin resistant AA and CA possess several differences in regards to the metabolic syndrome prevalence and associated factors, it is important to examine the responsiveness to interventions by ethnicity.

In the abstract to be presented in June 2007, we have intriguing results of differential hormonal responses to daily activities. We found that anthropometric measures were independent of the insulin response to a meal and that the insulin response was higher in AA. The hormonal profiles of AA differ from those in CA and appear to be similar to those seen in more obese individuals: lower morning cortisol levels and higher insulin concentrations in response to a meal. This exaggerated insulin response in healthy AA may be an early indication of pre-insulin resistance, a risk factor for cardiovascular disease and Type 2 diabetes mellitus. The relations among these hormonal differences, life stress and ethnicity deserve further exploration.

Our goals for the final year of the study include completion of testing participants, analyzing data, continuing biochemical and statistical analyses, and examining data for HPA reactivity, resistance to feedback control and insulin resistance, and exercise-associated increases in insulin and glucocorticoid sensitivity. We have successfully met our previous goals and are ahead of schedule in many areas. We have enrolled over 124 participants to date and have completed testing of 93 participants. We also submitted six abstracts and gave four poster presentations based on our physiological, biochemical, and psychological testing. In addition, we have prepared two manuscripts discussing the criteria of maximal aerobic testing in AA and the correlates of insulin resistance between AA and CA. Over the next year we will continue to examine differences between CA and AA in terms of potential underlying causes of the metabolic syndrome and how different physiologic stressors activate the HPA axis and metabolic processes intrinsic to obesity and associated CHD risk factors. We expect to add to the body of knowledge that surrounds the metabolic syndrome, because of our comparative design as a

function of ethnicity, weight, and gender. We will also continue to examine the data, prepare manuscripts, and prepare the final report on the results.

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APPENDICES

None